

# EFFECTS OF TYPE VI COLLAGEN ON MACROPHAGES

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Emphysema is an abnormal inflammatory response of the alveoli that lose their elasticity due to destruction of alveolar septi. Collagen, an extracellular matrix protein (ECM), is expressed in the lung, which is important in maintaining the integrity of the tissue. Destruction of the ECM components in the alveolar structure contributes to the development of emphysema. We have found that the gene expression of type VI collagen (*COL6A1*) is higher in the lungs of emphysema patients as compared to that from normal controls. Type VI collagen (COL6) is found in the pulmonary interstitial compartment where massive macrophages are infiltrated in the inflammatory environment. The hypothesis is that excessive COL6 activates macrophages to mediate inflammatory responses, which may contribute to the pathogenesis of emphysema. The goal is to define the effects of type VI collagen on macrophages. Results from murine bone marrow derived macrophages showed a marked increase in the numbers of CD86-positive cells after soluble COL6 stimulation. To further support the stimulatory function of COL6, human THP-1 cells as well as primary monocytes produced inflammatory cytokines IL-12 and IFN $\gamma$  following COL6 stimulation. Taken together, our data has demonstrated the stimulatory effects on macrophages by COL6 stimulation, which may mediate the inflammatory responses in the pathogenesis of emphysema.